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<input type="checkbox"/>	L7	L6 and l3	14
<input type="checkbox"/>	L8	L7 or l5	117
<input type="checkbox"/>	L9	L8 and (indolinones or \$linones).clm.	40
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<input type="checkbox"/>	L11	L10 and (normal\$ or control).clm.	11
<input type="checkbox"/>	L12	L11 and (indolinones or indolinone or \$linones).clm.	0
<input type="checkbox"/>	L13	L10 and (normal\$ or control).clm.	11
<input type="checkbox"/>	L14	L11 and (indolinones or indolinone or \$linones)	7
<input type="checkbox"/>	L15	l14 and l11	7
<input type="checkbox"/>	L16	l2 and measur\$.clm. and compar\$.clm. and detect\$.clm.	5
<input type="checkbox"/>	L17	l2 and (src or frk or btk or csk or abl or zap70 or zap-70 or fes or fps or fak or jak or ack or yes or fyn or lyn or lck or blk or hck or fgr or yrk or ras or raf).clm.	32

END OF SEARCH HISTORY

Dev Growth Differ. 2005 May;47(4):233-42.

Related Articles, Links

**Postnatal changes in the expression of p60c-Src in mouse testes.****Gye MC, Choi JK, Ahn HS, Kim YS.**

Department of Life Science, College of Natural Sciences, Hanyang University, Seoul 133-791, Korea. mcgye@hanyang.ac.kr

Src family non-receptor tyrosine kinases are involved in signaling pathways which mediate cell growth, differentiation, transformation and tissue remodeling in various organs. In an effort to elucidate functional involvement of p60c-Src (c-Src) in spermatogenesis, the postnatal changes in c-src mRNA and c-Src protein together with kinase activity and subcellular localization were examined in mouse testes. c-src mRNA levels in testes increased during the first 2 weeks of postnatal development (PND). Following a decrease at puberty (PND 28), the c-src mRNA levels re-increased at adulthood (PND 50). Src kinase activity of testes was low at PND 7 but sharply increased prepubertally (PND 15) and highest at adulthood. Upon Western blotting, the level of c-Src protein was the highest in prepubertal testes but rather decreased in adult testes at PND 50. In adult testes, ubiquitination of c-Src proteins was apparent compared with immature one at PND 7, suggesting active turnover of c-Src by ubiquitination. In immature testes, c-Src immunoreactivity was largely found in the cytoplasm of the Sertoli cells. By contrast, in pubertal and adult testes intense immunoreactivity was localized at the adluminal and basal cytoplasm of Sertoli cells bearing elongated spermatids and early germ cells, respectively. The immunoreactivity of c-Src in the Leydig cells was increased during pubertal development, suggesting the functional involvement of c-Src in differentiated adult Leydig cells. Throughout postnatal development, some spermatogonia and spermatocytes showed intensive c-Src immunoreactivity compared with other germ cells, suggesting a possible role of c-Src in germ cell death. Taken together, it is suggested that c-Src may participate in the remodeling of the seminiferous epithelia and functional differentiation of Leydig cells during the postnatal development of mouse testes.

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CSKS	17
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ZAP70	484
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- ☐ 1. [20050125852](#). 07 May 04. 09 Jun 05. Novel kinases. Caenepeel, Sean, et al. 800/18; 435/194 435/320.1 435/325 435/6 435/69.1 536/23.2 C12Q001/68 A01K067/027 C07H021/04 C12N009/12.
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The spleen tyrosine kinase (Syk) in human disease, implications for design of tyrosine kinase inhibitor based therapy.**Navara CS.**

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The spleen tyrosine kinase Syk is an enigmatic protein tyrosine kinase functional in a number of diverse cellular processes. It is best known as a non-receptor protein tyrosine kinase involved in signal transduction in cells of hematopoietic origin and plays a crucial role in signaling in most of these cells. It is involved in B and T-cell function, platelet aggregation, mast cell signaling, neutrophils and macrophages. Recently it has been found in tissues outside of the hematopoietic lineage. Perhaps the most interesting non-traditional role of Syk is that of a potential tumor suppressor in breast cancer. Absence of Syk protein in primary breast tumors is correlated with poor outcomes. Syk deficient cells have increased motility which is restored to normalcy by replacement with wild-type Syk. Syk also associates with the actin and tubulin cytoskeleton and is an alpha-tubulin kinase. The central role that Syk has in a number of cellular processes makes it an ideal starting point for broad therapeutic targeting.

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